

Baby 4 had 40 U/ml IgE at zero time and baby 5 had 180 U/ml at zero time. Since each weighed approximately 7 pounds at the time of transfusion, we may assume that the differences in establishing equilibrium between the serum and the cell-fixing sites is not due to dilution differences but rather is a function of the input IgE level and the total number of cell binding sites in the skin (and elsewhere).

It can be seen in Table III that both infants reached their maximum skin-fixed IgE level at about 2 days. However, the baby with 180 U/ml of IgE after transfusion had skin reactivity at a dilution of 1:50,000 of anti-IgE antiserum at the same time (12 hr after transfusion) that the other baby, who had 40 U/ml at zero time was skin reactive at a dilution of 1:5000.

A stable lower level of serum IgE concentration was seen by about 2 days (51 hr) in the baby (No. 5) with the higher input IgE. That is, starting at 180 U/ml, this baby stabilized at 10% (~18 U/ml) of input; baby 4, on the other hand, with a 40 U/ml input, took about 3½ days (84 hr) to level off at 20% of input (8 U/ml). Thus the baby with the lower input IgE took 65% longer to reach equilibrium in the serum and stabilized at less than half the serum IgE level of the high input baby (8 vs 20 U/ml). Both of these findings are confirmed by the reaction to anti-IgE-Fc in the skin. That is, the high-input baby (No. 5) reached a 1:50,000 titer by 12 hr, a level attained by baby 4 (as this baby's maximum) by 37 hr. The high-input baby continued to increase his skin-fixed IgE concentrations, reaching his peak of 1:100,000 at 51 hr.

With the addition of further data, the foregoing may provide the basic biologic parameters for understanding the kinetics of the equilibrium between serum IgE and cell-(skin) fixed IgE. An interesting question that remains to be answered is the effect of the infusion of large amounts of (adult) IgE in the newborn on that infant's synthesis of his own IgE. In one infant that we were able to follow for 31 days we noted the possible appearance of newly synthesized IgE about 8.7 days after exchange transfusion. If this observation is confirmed, it would suggest that the exchange transfusion prematurely stimulated the "turning-on" of the infant's IgE system rather than that the infusion of large amounts of IgE depressed the synthesis of IgE by the infant. Alternatively, the additional IgE could have derived from the donor's cells rather than the infant's.

In addition to providing information on the fate of passively administered adult human IgE to a normal human newborn, these data confirm the potential anaphylactic hazard of the prolonged sensitization by IgE received in transfusions, as first described in JAMA in 1919 by M. A. Ramirez.

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Occupational allergy secondary to inhalation of garlic dust

To the Editor:

The article by Falleroni et al.¹ presented a clinical case of an atopic patient who suffered from asthma when he inhaled garlic dust. In 1980 Couturier et al.² described two similar clinical cases in atopic patients.

An 11-yr-old boy living on a farm had severe asthma every July for 3 yr when garlic bulbs were collected. This boy was accustomed to helping his parents when the crop was harvested. The diagnosis of allergy to garlic was made by a positive scratch test, a positive basophil degranulation test,³ and a bronchial provocation test with garlic dust showing a decrease of FEV₁ within 15 min of 35% of the initial values. The extract of garlic was made from the skin of the clove, in contrast to the methods of Falleroni et al.,¹ who used the whole garlic grain. Precipitins against garlic dust were absent.

The second case was that of a 15-yr-old boy who helped a farmer during the collection and storage of garlic. He presented with asthma, rhinitis, and conjunctivitis from July through November. Positive scratch and basophil degranulation tests confirmed the diagnosis.

Since the publication of the paper by Couturier et al.,² three other cases of occupational garlic allergy have been documented in the same area, demonstrating that allergy to garlic dust has to be considered in areas where garlic is cultivated or stored. Moreover, in the French publication the extracts were made from the garlic skin instead of the whole clove. Finally it was shown in both articles^{1, 2} that garlic dust is able to induce an immediate-type allergic reaction when inhaled; this reaction was found to be of reaginic origin¹ without the participation of precipitins.²

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